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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/063,517	05/01/2002	Dan L. Eaton	P3230R1C001-168	8146

30313 7590 03/31/2005

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EXAMINER	
SAOUD, CHRISTINE J	
ART UNIT	**PAPER NUMBER**
1647	

DATE MAILED: 03/31/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/063,517	EATON ET AL.	
	Examiner	Art Unit	
	Christine J. Saoud	1647	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *18 January 2005*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-5* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-5* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *011805*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Claim 1 has been amended and claim 6 has been canceled in the paper filed 18 January 2005. Claims 1-5 are pending in the instant application and under examination.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Any objection or rejection of record which is not expressly repeated in this action has been overcome by Applicant's response and withdrawn.

Applicant's arguments filed 18 January 2005 have been fully considered but are not deemed persuasive.

Inventorship

In view of the papers filed 18 January 2005, the inventorship in this nonprovisional application has been changed by the deletion of Dan L. Eaton, Ellen Filvaroff, Mary E. Gerritsen, and Colin K. Watanabe.

The application will be forwarded to the Office of Initial Patent Examination (OIPE) for issuance of a corrected filing receipt, and correction of Office records to reflect the inventorship as corrected.

Specification

Applicant's submission of a substitute specification is noted.

37 CFR § 1.125 Substitute specification.

(a) If the number or nature of the amendments or the legibility of the application papers renders it difficult to consider the application, or to arrange the

> papers for printing or copying, the Office may require the entire specification, including the claims, or any part thereof, be rewritten.
>
> (b) Subject to § 1.312, a substitute specification, excluding the claims, may be filed at any point up to payment of the issue fee if it is accompanied by a statement that the substitute specification includes no new matter.
>
> (c) A substitute specification submitted under this section must be submitted with markings showing all the changes relative to the immediate prior version of the specification of record. The text of any added subject matter must be shown by underlining the added text. The text of any deleted matter must be shown by strike-through except that double brackets placed before and after the deleted characters may be used to show deletion of five or fewer consecutive characters. The text of any deleted subject matter must be shown by being placed within double brackets if strike-through cannot be easily perceived. An accompanying clean version (without markings) must also be supplied. Numbering the paragraphs of the specification of record is not considered a change that must be shown pursuant to this paragraph.

The substitute specification filed 18 January 2005 has not been entered because it does not conform to 37 CFR 1.125(b) and (c) because: Applicant has failed to provide a statement that the substituted specification includes no new matter and because a marked up copy showing the changes relative to the immediate prior version of the specification is not provided. It is noted that the "substitute specification" has omitted the first paragraph of the specification indicating the claim of priority.

The disclosure is objected to because it contains an embedded hyperlink and/or other form of browser-executable code. Applicant is required to delete the embedded hyperlink and/or other form of browser-executable code. See MPEP § 608.01. See at

least page 31, paragraph [0205] and page 35, paragraph [0216] (relative to the original specification).

Claim Rejections - 35 USC §§ 101/112

Claims 1-5 stand rejected under 35 U.S.C. 101 because the claimed invention is not supported by either a specific and substantial asserted utility or a well established utility for the reasons of record in the previous Office action and for those reasons provided below.

Applicant argues at page 7 of the response that "the gene encoding the PRO300 polypeptide is differentially expressed in certain cancers compared to normal tissue and is useful as a diagnostic tool". Applicant's arguments are not persuasive because the data provided in the instant specification does not support this conclusion. As stated in the previous Office action, Example 18 at page 140 and 141 of the specification indicates that the polynucleotide of SEQ ID NO:12 is "more highly expressed" in "normal" lung as compared to lung tumor tissue. However, there is no guidance on how to use this information because no details regarding expression levels (relative or absolute) are disclosed. The information provided is too sparse to allow the encoding polynucleotide to be used as a diagnostic marker for lung tumors. The claims are directed to the antibody (useful for detecting the protein) and the specification provides no information regarding protein expression, so there is no basis to conclude that the protein and/or antibody could be used as a diagnostic tool, absent evidence to the contrary. Because it is not known if the nucleic acid is involved in tumor suppression or

if it is inhibited by tumor progression, the skilled artisan could not use it therapeutically as a target for treatment of a tumor or as a therapeutic for treatment of a tumor. As stated before, there is no information regarding the polypeptide and its relation to cancer (or lack thereof), so the skilled artisan could not use it or the antibody that binds it therapeutically or diagnostically as asserted by Applicant. Therefore, Applicant's conclusion that the claimed invention "is useful as a diagnostic tool" is not supported by any facts of record.

Applicant has submitted two Declarations by Dr. Grimaldi (exhibits 1 and 2), one Declaration by Dr. Polakis (exhibit 3) and one Declaration by Dr. Ashkenazi (exhibit 5). The Declarations under 37 CFR 1.132 filed 18 January 2005 are insufficient to overcome the rejection of the claims based upon lack of utility as set forth in the last Office action for the reasons provided below.

Applicant asserts that the Grimaldi (1) Declaration indicates that "any visually detectable difference seen between two samples is indicative of at least a two-fold difference in cDNA between the tumor tissue and the counterpart normal tissue" and that "the results of the gene expression studies indicate that the genes of interest 'can be used to differentiate tumor from normal'". One skilled in the art would not conclude that such a weak correlation would indicate that PRO300 is a diagnostic marker for lung cancer for the following reasons. The art recognizes that lung epithelium is at risk for cellular damage due to direct exposure to environmental pollutants and carcinogens, which result in aneuploidy *before* the epithelial cells turn cancerous. See Hittelman (2001, Ann. N. Y. Acad. Sci. 952:1-12), who teach that damaged, *pre-cancerous* lung

epithelium is often aneuploid. See especially p. 4, Figure 4. Because aneuploid DNA can be found in normal tissue, detection of increased DNA copy number does not necessary mean those cells containing the DNA are cancerous. The gene amplification assay disclosed in the instant specification does not provide a comparison between the lung tumor samples and normal lung epithelium control, and thus it is not clear that PRO300 is amplified in cancerous lung epithelium more than in damaged (non-cancerous) lung epithelium. Thus, one skilled in the art could not conclude that PRO300 is a diagnostic probe for lung cancer based on the evidence provided. Furthermore, the claims are directed to antibodies which bind the encoded polypeptide, and the specification provides no evidence or data to suggest that the encoded polypeptide has any relation to lung cancer because there is no information on the expression of the encoded polypeptide.

Additionally, Example 18 of the specification used "one or tumor tissues" to determine cDNA levels of the gene of interest. However, it is common for different types of cancer of a particular tissue or organ to have different gene expression patterns. For example, different types of breast cancer demonstrate different gene expression patterns and the measurement of a single gene would not necessarily be indicative of the presence or absence of cancer (i.e. not diagnostic). Since the specification fails to indicate what types of lung tumor tissue was used in the experiment, one of ordinary skill in the art would not be able to make a reasonable assessment of the results of PCR of the disclosed polynucleotide because not all lung tumor tissue would be expected to have the same expression pattern for the disclosed

gene, absent evidence to the contrary. The data presented in the micro array assay are preliminary at best, and cannot be evaluated or repeated independently by the skilled artisan, and the asserted use of the claimed invention as a diagnostic marker or as a therapeutic target is merely an invitation to experiment.

At page 8 of the response, Applicant states "the accepted understanding in the art is that there is a direct correlation between mRNA levels and the level of expression of the encoded protein" and that the Grimaldi (2) Declaration states "in the vast majority of cases, when a gene is over-expressed ... the gene product or polypeptide will be over-expressed .... This same principal applies to gene under-expression.". These statements have no basis in fact and appear to be the opinion of the Declarant.

The declaration by Dr. Grimaldi has been fully considered but is not deemed persuasive. At paragraph 4, the Declarant discusses mutations of Her2/Neu, and chromosomal translocations that are known to be associated with cancer, and states that "If the chromosomal aberration results in the aberrant expression of a mRNA and the corresponding gene product (the polypeptide) as they do in the aforementioned cases, then the gene product is a promising target for cancer therapy, for example, by the therapeutic antibody approach." This argument has been fully considered but is not deemed persuasive because it evidences that the instant specification provides a mere invitation to experiment, and not a readily available utility. The PRO300 gene, unlike Her2/Neu, has *not* been associated with tumor formation or the development of cancer, nor has it been shown to be predictive of such. Similarly, unlike t(5;14), no translocation of PRO300 is known to occur. All that the specification demonstrates is that the

PRO300 nucleic acid was amplified uncharacterized lung tumor tissue and no information as to relative fold increase is provided. No mutation or translocation of PRO300 has been associated with lung tumor tissue. It is not known whether PRO300 is expressed in normal lung tissue or in lung tumor tissue, and what the relative levels of expression are. In the absence of any of the above information, all that the specification does is present evidence that the DNA encoding PRO300 is amplified in an undisclosed number of samples, and invite the artisan to determine the rest of the story. Such is insufficient to meet the requirements of 35 U.S.C. §101 for the claimed invention.

At paragraph 5 of the declaration, Declarant argues that increased mRNA expression is expected to be associated with increased protein production. This argument has been fully considered but is not deemed persuasive because (a) this appears to be Declarant's opinion, and is not supported by fact or evidence (b) there has been no distinction on the record in general or in the specification as filed between total nucleic acid, which includes chromosomal DNA, and mRNA. One cannot determine from the data in the specification whether the observed "amplification" of nucleic acid is due to increase in chromosomal copy number, or alternatively due to an increase in transcription rates. It remains that there is no information on the record as to whether the encoded protein is expressed *at all* in lung tissue, cancerous or otherwise. The literature reports that it does not necessarily follow that an increase in gene copy number results in increased gene expression and increased polypeptide expression, contrary to Applicant's and Declarant's assertions. For example, Pennica et al. (1998, PNAS USA 95:14717-14722) disclose that:

> "An analysis of *WISP*-1 gene amplification and expression in human colon tumors showed a correlation between DNA amplification and overexpression, whereas overexpression of *WISP*-3 RNA was seen in the absence of DNA amplification. In contrast, *WISP*-2 DNA was amplified in the colon tumors, but its mRNA expression was significantly reduced in the majority of tumors compared with the expression in normal colonic mucosa from the same patient."

It remains that, as evidence by Pennica et al., the issue is simply not predictable, and the specification presents a mere invitation to experiment. This is further borne out by paragraph 6, which proposes further experimentation, should Applicants assertions be erroneous.

At page 8 of the response, Applicant presents a declaration by Dr. Polakis (3) filed with the response under 37 CFR 1.132. In the declaration, Dr. Polakis states that the primary focus of the Tumor Antigen Project was to identify tumor cell markers useful as targets for cancer diagnostics and therapeutics. Dr. Polakis states that approximately 200 gene transcripts were identified that are present in human tumor cells at significantly higher levels than in corresponding normal human cells. Dr. Polakis states that antibodies to approximately 30 of the tumor antigen polypeptides have been developed and used to show that approximately 80% of the samples show correlation between increased mRNA levels and changes in polypeptide levels. Dr. Polakis states that it remains a central dogma in molecular biology that increased mRNA levels are predictive of corresponding increased levels of the encoded polypeptide. Dr. Polakis characterizes the reports of instances where such a correlation does not exist as exceptions to the rule. This has been fully considered but is not found to be persuasive. First, it is important to note that the instant specification provides no information

regarding increased mRNA levels of PRO300 in tumor samples relevant to normal samples. Only gene amplification data was presented in Example 18; no information is provided regarding protein expression levels. Therefore, the declaration is insufficient to overcome the rejection of the claims based upon 35 U.S.C. §§ 101 and 112, first paragraph, since it is limited to a discussion of data regarding the correlation of mRNA levels and polypeptide levels, and not gene amplification levels and polypeptide levels. Furthermore, the declaration does not provide data such that the examiner can independently draw conclusions. Only Dr. Polakis' conclusions are provided in the declaration. There is no evidentiary support to Dr. Polakis' statement that it remains a central dogma in molecular biology that increased mRNA levels are predictive of corresponding increased levels of the encoded polypeptide. Finally, it is noted that the literature cautions researchers from drawing conclusions based on small changes in transcript expression levels between normal and cancerous tissue. For example, Hu et al. (2003, Journal of Proteome Research 2:405-412) analyzed 2286 genes that showed a greater than 1-fold difference in mean expression level between breast cancer samples and normal samples in a micoarray (page 408, middle of right column). Hu et al. discovered that, for genes displaying a 5-fold change or less in tumors compared to normal, there was no evidence of a correlation between altered gene expression and a known role in the disease. However, among genes with a 10-fold or more change in expression level, there was a strong and significant correlation between expression level and a published role in the disease (see discussion section).

Also Gygi et al. (Mol. Cell. Biol. 19(3): 1720-1730, 1999) analyzed 156 genes from *S. cerevisiae* to study the correlation between protein and mRNA abundance. Gygi et al. concluded that transcript levels provide little predictive value with respect to the extent of protein expression (see page 1730, column 1, final sentence). This conclusion stems from the data presented in Figure 5 of Gygi et al. Although there was a general trend of increased protein levels resulting from increased mRNA levels, Gygi et al. state that this number was highly biased by a small number of genes with very large protein and message levels (see page 1726, column 1, paragraph 2). For genes with low message levels (69% of the genes studied), the correlation coefficient was only .356, demonstrating that message levels are generally not predictive of protein levels for the majority of the genes studies by Gygi et al.

Applicant cites statements from a text book (Molecular Biology of the Cell) regarding gene expression and protein expression (page 12 of the response). Applicant specifically points to Figure 6-3 as supporting the conclusion that increased gene expression correlates to increased protein expression. However, the evidence of the studies by researchers in the field contradict this conclusion (see above). Furthermore, the text provided by Applicant at page 379 demonstrates that there are many other factors which play a role in the pathway of DNA to RNA to protein, and control is exerted at a number of different levels in a number of different ways, which would explain why there is no established direct correlation between gene expression levels and protein expression levels. The system is obviously much more complex than the simplistic diagram referenced by Applicant in Figure 6-3 and there is no evidence in the

reference which supports the conclusion that there is a direct correlation between the level of mRNA and the level of the encoded protein, and this assertion is contradicted by the experimental evidence of Hu et al., Gygi et al., and Pennica et al.

Applicant argues at page 9 that the claimed antibodies would have diagnositic utility even if there is no direct correlation between gene expression and encoded polypeptide. At page 10 of the response, Applicant refers to the Grimaldi (2) declaration for stating "if over- or under-expression of a gene product does not correlate with over- or under-expression of mRNA in certain tumor types but does so in others, then identification of both gene expression and protein expression enables more accurate tumor classification and hence better determination of suitable therapy". These arguments are not persuasive. First, the utility of the encoded polypeptides (and therefore, the antibodies which bind them) on their face has been addressed above. As stated previously, the data and evidence in the specification is too sparce for one of ordinary skill in the art to make any reasonable conclusions as to the utility of the encoded polypeptides and their antibodies for diagnostic purposes because only the cDNA was measured, because of aneuploidy, because no information is provided on expression levels of the protein, because there is no information on what type of lung tumor tissues were tested, etc. Secondly, the statements of Declarant in paragraph #6 in exhibit 2 demonstrate that the invention is not complete and cannot be used without further experimentation (i.e. identification of both gene expression and protein expression; again, there is no protein expression data at all in the instant specification).

At page 9 of the response, Applicant refers to the declaration of Dr. Ashkenazi as

indicating that simultaneous testing of gene expression and gene product enables more accurate tumor classification, even if there is no positive correlation between the two. The Declaration of Dr. Ashkenazi explains that even when amplification of a cancer marker gene does not result in significant over-expression of the corresponding gene product, this very absence of gene product over-expression still provides significant information for cancer diagnosis and treatment, in that if the gene product is over-expressed in some tumor types but not others, this would enable more accurate tumor classification and hence better determination of suitable therapy, and additionally, if a gene is amplified by the corresponding gene product is not over-expressed, the clinician accordingly will decide not to treat a patient with agents that target that gene product

The declaration filed under 37 CFR 1.132 filed 18 January 2004 is insufficient to overcome the rejection of the claims based upon lack of utility as set forth in the last Office action because: it has not been demonstrated that the protein of the instant invention is differentially expressed in different tumors. If it was, the protein would have a specific and substantial utility for tumor classification, but the mere assertion that it may be differentially expressed does not provide a specific and substantial utility, and is an invitation to experiment. The argument that if a gene is amplified but the gene product is not over-expressed, the clinician accordingly will decide not to treat a patient with agents that target the gene product is also insufficient to overcome the rejection of the claims. If a specific gene product was known to be involved in cancer and if there were known compounds that could be used to target the gene product, this would be an acceptable utility. However, the gene product of the instant invention has not been

demonstrated to be involved in cancer. Over-expression of a gene product in a cancer cell does not necessarily mean that the gene product is involved in the cancer and that targeting the gene product would be therapeutic. Additionally, there are no known compounds that would target the gene product.

Applicant provides the Hanna et al. reference to support the Declaration of Dr. Ashkenazi. The Hanna reference is not applicable to the instant fact situation, as it deals with a known tumor associated gene, and not with a prospective analysis of the type found in this specification.

At page 11 of the response, Applicant asserts that "there are significant data that show that the gene encoding the PRO300 polypeptides is more highly expressed in normal lung tissue compared to lung tumor" and that these "data are strong evidence that the gene encoding the PRO300 polypeptide is associated with lung tumors". This argument is not persuasive for the reasons presented above. Furthermore, there are a multitude of genes that are expressed in tissues and their expression patterns may change depending on the disease state of the tissue being studied, but this alone does not implicate them as being associated with the disease state, absent evidence to the contrary. The data presented in the micro array assay are preliminary at best and cannot be evaluated or repeated independently by the skilled artisan. The data presented in Example 18 indicate nothing about the encoded polypeptide or the antibodies that bind the encoded polypeptide. The proposed use of the claimed invention is simply a starting point for further research and investigation into potential practical uses of the claimed invention.

For these reasons, there is no substantial and specific utility for the antibodies that bind the polypeptide of SEQ ID NO:12.

Claims 1-5 are also rejected under 35 U.S.C. 112, first paragraph. Specifically, since the claimed invention is not supported by either a specific and substantial asserted utility or a well established utility for the reasons set forth above, one skilled in the art clearly would not know how to use the claimed invention for the reasons of record.

Applicant asserts that the arguments regarding the utility of the claimed invention are sufficient to overcome the enablement rejection of the claims under 35 U.S.C. 112. However, since the submitted arguments were not persuasive to overcome the utility rejections under 35 U.S.C. 101, they are also not persuasive to overcome the instant rejection.

It would require significant further experimentation to be able to use the claimed antibodies because no definite function has been determined for the encoded protein and there is no definite function supported by the prior art. The specification does not provide sufficient guidance or working examples to be able to use the encoded polypeptide nor the antibodies that bind it diagnostically or therapeutically, for example in association lung tumors, without undue experimentation.

35 U.S.C. § 102

The following rejections under 35 U.S.C. § 102 is made under the assumption that the effective filing date for the instantly claimed invention is 05/07/2002, which is the actual filing date of the instant application. Applicant is advised that the instant application can only receive benefit under 35 U.S.C. §120 from an earlier application which meets the requirements of 35 U.S.C. § 112, first paragraph, with respect to the new claimed invention. Because the instant application does *not* meet the requirements of 35 U.S.C. § 112, first paragraph, for the reasons given above and it is a continuing application of Serial Number 10/006,867, the prior application also does not meet those requirements for the claimed invention and, therefore, is unavailable under 35 U.S.C. § 120.

Claim Rejections - 35 USC § 102

Claims 1-5 stand rejected under 35 U.S.C. 102(b) as being anticipated by WO 01/16318 for the reasons of record in the previous Office action.

Applicant argues that they have made a proper claim of priority under 120 to obtain benefit of the WO 01/16318. However, benefit under 120 requires fulfillment of the requirements of 112, which includes a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same, and shall set forth the best mode contemplated by the inventor of carrying out his invention. Since the instant

specification does not meet the requirements of 35 U.S.C. 112, the instant application does not obtain benefit of the earlier filed application. Since the earlier filed application was published more than 1 year before filing the instant application, it is proper art under 102(b).

Applicant argues that the data in Example 18 was first disclosed in PCT application PCT/US00/23328, and therefore, priority benefit should date back to this application. However, because of the reasons of record, the data in this example does not provide a specific, substantial and credible utility for the claimed invention, and therefore, the requirements of 35 U.S.C. 112, first paragraph are not met and benefit is not granted.

Conclusion

No claim is allowed.

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of

the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Christine J. Saoud whose telephone number is 571-272-0891. The examiner can normally be reached on mttr, 8:00-2:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Brenda Brumback can be reached on 571-272-0961. The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

CHRISTINE J. SAOUD
PRIMARY EXAMINER
Christine J. Saoud